

## A Case of Back Abscess and Diabetic Ketoacidosis in a Patient with Type 2 Diabetes Mellitus

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### Abstract

A 41-year-old woman with type 2 diabetes mellitus (DM) presented with diabetic ketoacidosis (DKA) and back abscess. Onset of complications was triggered by infection with methicillin-sensitive *Staphylococcus aureus* and the neglected hyperglycemic state. The patient responded to immediate incision and drainage and antibacterial therapy. Prompt surgical intervention, antibacterial therapy, and rapid restoration of glycemic control are crucial to prevent mortality in DM patients complicated with abscess of skin and soft tissues.

**Keywords:** Diabetes mellitus; Diabetic ketoacidosis; Back abscess; *Staphylococcus aureus*; Insulin

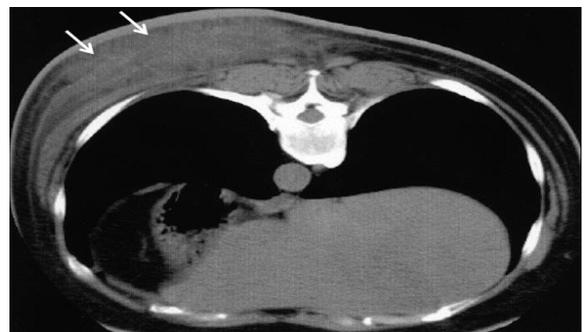
### Introduction

Patients with type 2 diabetes mellitus (DM) who have poor glycemic control are susceptible to severe bacterial infections and complications such as diabetic ketoacidosis (DKA), which may in turn further exacerbate the bacterial infection [1,2]. Commonly reported bacterial infections in DM patients in Japan have included infections of the respiratory system, kidney and urinary tract, and skin and soft tissue [2]. Deresinski reported that DKA is precipitated or complicated by infections in 75% of the cases. The author also reported that the mortality rate of patients with DKA and infections is 43% [3,4]. However, recent studies have documented a fall in mortality rates in patients with DKA and infections (0%-5%) [5-8]. A recent retrospective cohort study of patients with DKA and sepsis in Taiwan reported an in-hospital mortality rate of 5.6% (9/160) [9]. However, there are few reports on patients with complications of DKA and back abscess [1,10-12].

Here, we report a case of giant back abscess and DKA in a patient with type 2 DM, which readily responded to prompt surgical intervention and antibacterial therapy.

### Case Report

A 41-year-old woman presented with fever and back pain. The pain was in the left back region, continuous, and slightly relieved in the prone position, and it worsened over time. There was no family history of diabetes mellitus and no history of smoking or alcohol intake. At the time of admission, her blood pressure was 112/56 mmHg, pulse rate was 96 beats/min, temperature was 38.2°C, and respiratory rate was 20 breaths/min. The patient was alert and conscious; there was no conjunctival pallor, and auscultation revealed no cardiovascular or respiratory abnormalities. Her height, weight, and body mass index were 158 cm, 61.2 kg, and 24.5, respectively. On examination, a 20 cm tender inflamed area with ulceration was observed on the left side of the back (Figure 1). Laboratory investigation results were as follows: glucose (post-prandial) 603 mg/dL; glycated hemoglobin (HbA1c) 13.7%; plasma osmotic pressure 287 mOsm/L; lactic acid 7.9 mg/dL; Na<sup>+</sup> 117 mEq/L; K<sup>+</sup> 5.8 mEq/L; Cl<sup>-</sup> 78 mEq/L; white blood cell count (WBC) 23,900/μL (neutrophils 91.5 %); C-reactive protein (CRP) 26.6 mg/dL; hemoglobin 15.3 g/dL; platelet count 28.3 × 10<sup>4</sup>/μL; total proteins 6.8 g/dL; AST 16 IU/L; ALT 14 IU/L; LDH 289 IU/L; total cholesterol 200 mg/dL; triglycerides (post-prandial) 177 mg/dL; blood urea nitrogen 30.4 mg/dL; and creatinine 0.77 mg/dL. Spot urine test revealed proteinuria (urine protein 1+), glycosuria (urine sugar 4+), and ketonuria (dipstick urine ketone bodies 3+).



**Figure 1:** Plain computed tomography (CT) revealed a hypodense/isodense abscess with indistinct border on the left side of the back. Image taken in the prone position.

Blood gas analysis revealed metabolic acidosis with respiratory compensation (pH 7.36, PO<sub>2</sub> 99.3 mmHg, PCO<sub>2</sub> 19.9 mmHg, HCO<sub>3</sub><sup>-</sup> 14.3 mEq/L, base excess -5.7 mEq/L, anion gap 24.7 mEq/L). Normal lactic acid levels, absence of renal dysfunction, and presence of ketone bodies in urine indicated DKA. Neglected hyperglycemia was considered the prima facie trigger for DKA.

The patient was hospitalized with a diagnosis of DKA, and back abscess incision-drainage and debridement of left back was performed. Antibacterial therapy (Ampicillin 6 g/day and Clindamycin 1200 mg/day) was initiated.

Administration of saline hydration and continuous insulin infusion improved the acidosis the day after hospital admission and decreased the blood glucose levels to 250 mg/dL. Negative anti-glutamic acid decarboxylase antibodies and urine C-peptide immunoreactivity (92-151 μg/day) was suggestive of type 2 DM. Blood culture was negative; however, methicillin-sensitive *Staphylococcus aureus* (MSSA) was detected on pus culture. Antibiotic therapy was continued for 14

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days. Three days after admission, administration of saline hydration and transvenous continuous insulin infusion was discontinued. Intensive control of blood glucose was achieved with subcutaneous injection of short-acting insulin before each meal and intermediate-acting insulin at bedtime. Dietary intervention and patient education for self-administration of insulin injection were done. The size of the back swelling decreased (Figure 2), and the inflammatory findings resolved with treatment. No evidence of retinopathy, neuropathy, or nephropathy was observed. The patient was discharged after 21 days of treatment in the hospital. Her treatment plan after discharge was as follows: blood glucose control with self-administration of subcutaneous insulin injection, and regular follow-up of back pain as an outpatient.

## Discussion

Patients with DM are susceptible to infection because of decreased migratory ability of neutrophils, decreased phagocytic activity, impaired humoral immunity, increased adherence of microorganisms to diabetic cells, neuropathy, and microangiopathy [1-3,11,13]. Moreover, frequency of medical intervention in DM patients is a risk factor for bacteremia; therefore, these patients are particularly vulnerable to infections. Furthermore, complications of DKA can inhibit leukocyte function and aggravate infection.

A literature search for similar cases in Japan (excluding iatrogenic cases [14]) revealed seven cases of DKA with subcutaneous abscess of the back (Table 1). Most of these were type 2 DM patients with poor compliance to treatment being the trigger for DKA. Pancreatic DM is unlikely to result in DKA as compared to type 1 or type 2 DM because it is also associated with decreased glucagon secretion, which

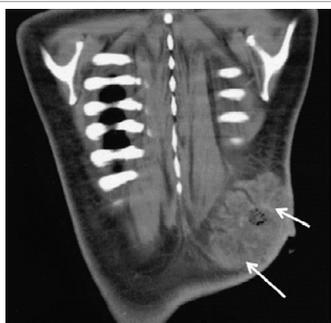


Figure 2: One week after the start of treatment the abscess had shrunk in size (coronal plane).

induces decreased production of ketone bodies [15]; however, cases of pancreatic DM-induced DKA have been reported [11]. Lankisch et al. [15] reported cases of chronic pancreatitis (pancreatic DM) with complications of splenic abscess and DKA.

Many of these patients had severe inflammation with WBC counts in excess of 20,000/ $\mu$ L (5/7 patients), CRP levels of 23.6-46.9 mg/dL, and HbA1c level of 11.7-15.1 (reference range: 11.3-14.7, according to the Japan Diabetes Society). Although MSSA is often the pathogenic bacterium that causes abscesses, cases of methicillin-resistant *S. aureus*-related abscesses have also been observed [1]. DM patients may be less able to defend against infection from *S. aureus* [10,12,16]. Joshi et al. [13] classified infections of the skin and soft tissue in DM patients into 2 types: mixed infections of Gram-negative bacilli and anaerobic bacteria and infections of Gram-positive cocci, such as *S. aureus* and hemolytic streptococci. The latter is more common in patients who have DKA complications and abscesses of the back. Furthermore, in patients with abscesses, DM-induced microangiopathy is believed to stimulate the growth of anaerobic bacteria because of the lack of oxygen in the peripheral tissue and decreased availability of antibacterial agent at the site of inflammation [13].

In abscesses of the skin and soft tissue, early surgical treatment will improve prognosis in DM patients. In the present case, incisional drainage and debridement were also performed early, and the massive abscess was successfully decreased in size. Common complications of surgical procedures are bleeding, bacteremia, leak of pus to abdominal or thoracic cavity, etc. There were no complications in our case. In the seven reported cases of DKA and back abscess, incisional drainage and/or debridement were performed in 7/8 cases. No deaths occurred among these seven cases.

We reported this DM case with complications of back abscess and DKA because prompt surgical procedure in addition to administration of antibacterial agents is crucial in decreasing mortality in DM patients complicated with abscess of skin and soft-tissues [13]. Moreover, most of these reported cases were patients with type 2 DM in which self-discontinuation of treatment or lack of treatment for hyperglycemia triggered DKA and back abscess. Therefore, patient education on the importance of treatment compliance and maintenance of glycemic control is a key tenet of treatment of DM.

## Acknowledgment

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Case	Year	Age (Years)	Gender	WBC ( $\mu$ L)	CRP (mg/dL)	HbA1c (%) (NGSP)	Types of diabetes	Background	Causative Organisms	Ref
1	2003	50	M	1500	23.6	13	Type 2	Self-discontinuation of treatment	MRSA, <i>Escherichia coli</i>	[1]
2	2003	40	F	46600	23.8	13.2	Type 2		<i>S. aureus</i>	[10]
3*	2005	22	F	27900	31.5	15.1	Type 2	drink-induced induced ketoacidosis		
4*	2006	47	M	24900	28.9	14	Type 2	Neglecting to treat a Hyperglycemic agent		
5	2008	51	M	25900	46.9	11.7	Pancreatic diabetes	Self-continuation of treatment	<i>S. aureus</i> , <i>Streptococcus</i> Spp.	[11]
6	2008	32	M	17000	?	13.6	Type 2		Anaerobic bacillus	[12]
7*	2012	60	M	?	?	12.2	Type 2	Self-discontinuation of treatment	MSSA	
8	Our case	41	F	23900	26.6	13.7	Type 2	Neglecting to treat a hyperglycemic state agent	MSSA	

F: Female; M: Male; NGSP: National Glycohaemoglobin Standardization Program; *S. aureus*: *Staphylococcus aureus*; MRSA: Methicillin-resistant *Staphylococcus aureus*; \*Japanese proceeding.

Table 1: Characteristics of patients with diabetic ketoacidosis and back abscess.

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